

pulmonary area. Contrary to what is usually the case in congenital heart lesions, cyanosis and clubbing of the fingers do not occur.

Five cases have come under Gibson's observation during the last few years. One of these he reports in detail, to substantiate his statement that cases of uncomplicated patent ductus arteriosus can be readily diagnosed. The main points in this case were as follows: The patient was a woman, aged thirty-one years. There was nothing of importance in the family history. She had always been subject to palpitation on exertion or excitement. There was no clubbing of the fingers, but a slight bluish color to the lips. The pulse was usually regular and equal in force and rhythm. There was slight bulging of the second, third, and fourth left costal cartilages, especially the second, close to the sternum. The point of maximum cardiac impulse was in the fifth interspace three and a half inches from the sternum. The right border of the heart reached one and a half inches and the left four and a half inches from the midsternum. There was a thrill over the base of the heart. It was felt with greatest intensity in the third left intercostal space, two and a half inches from the midsternum. It began shortly after the apical impulse and continued almost to the next apex beat. The shock of the pulmonary second sound could be distinctly felt during its continuance. At the base of the heart an almost continuous rushing murmur, beginning shortly after the first sound, which was clear at its commencement, and continuing almost to the beginning of the next first sound, was heard. It had its maximum intensity in the third left interspace, about two and a half inches from the midsternal line, but was heard all over the base of the heart. The second sound was heard distinctly in the middle of this murmur; it was distinctly accentuated and sometimes doubled. The sounds at the other areas were all audible, some being more obscured than others by the transmitted murmur.

With a thrill and murmur beginning after the first sound and continuing until after the end of the second sound, there was no possibility entertained in Gibson's mind that the physical signs were due to an alveolar lesion. It seemed to him that no other diagnosis was possible than patency of the ductus arteriosus.

The patient at a subsequent date was admitted with acute lobar pneumonia, from which she eventually died. An autopsy was obtained and Gibson's diagnosis of patent arterial duct was substantiated. The ductus was so short that at first sight it seemed as if the left pulmonary artery was actually adherent to the aorta. The duct admitted a No. 12 to 14 bougie. There were no abnormal communications between the various heart cavities. The various valves were normal; the tricuspid orifice was dilated, however. The right ventricle was dilated and hypertrophied, the wall measuring three-sixteenths of an inch in thickness.

Gibson states that this case shows that in certain instances the diagnosis must in large part be made "from the stand-point of clinical observation, instead of from the consideration of the pathological changes in the heart and the resulting mechanical disturbances."

The writer sums up the essential facts upon which the diagnosis of persistent ductus arteriosus may be made with perfect confidence as follows:

There may be no dyspnoea, cyanosis, œdema, or other evidence of disturbance of the general circulation. Inspection may fail to show anything of importance. Palpation usually reveals the long thrill following the apical impulse and persisting beyond the recoil of the blood on the semilunar cusps, which may be felt during the thrill. Percussion may not show any enlargement of the heart. Auscultation gives convincing evidence of the lesion in a murmur which may be regarded as almost pathognomonic. Beginning distinctly after the first sound, it accompanies the latter part of that sound, which may be accentuated in the pulmonary area, or may be, and often is, doubled, and finally dies away during the long pause. This murmur is best heard in the second or third left intercostal spaces.

Concerning the Cause of the Tolerance to Morphine.—According to FAUST, the tolerance of the organism to increasing doses of morphine might appear to be possible of explanation in two ways:

1. It may be due to a dulling of the nervous system against the action of the drug, so that given doses eventually may not have as marked an effect as at first. Such an effect would be expected to occur only after large doses.
2. The organism gradually comes to possess the power of rendering the morphine ineffectual by transforming or destroying it.

By experimentation on dogs with increasing doses of the drug he comes to the conclusion that the latter explanation is apparently the correct one. He finds that under normal conditions a portion of the morphine injected subcutaneously or intravenously is transformed in the body tissues. After a single dose of morphine, sufficient to produce definite poisonous symptoms in dogs, he was able to obtain only about 70 per cent. of the amount injected. By gradually increasing the size of the injected dose a stage is reached when not a particle of morphine is recoverable in the feces or urine. Further, an analysis of the various organs fails to show the presence of any morphine. During the experiments the reduction in the percentage of recoverable morphine in the feces was gradual, until eventually none was found.

Faust concludes from his investigations that the tolerance of the organism to morphine is due not to a tolerance or a dulling of the tissues to the effect of the drug, but rather to the increasing power which the tissues come to possess to transform and destroy the alkaloid and thus render it inactive.—*Ueber die Ursachen der Gewöhnung an Morphin*. Edwin Stanton Faust. Leipzig: Druck von J. B. Hirschfeld, 1900.

Change in the Pathology of Influenza.—WASSERMANN (*Deutsche med. Wochenschrift*, 1900, No. 28) calls attention to some observations made in the Institute for Infectious Diseases, according to which, in the influenza cases with pneumonia observed last year, the influenza bacilli were found in very scanty numbers, and soon disappeared. Notwithstanding this, severe toxic symptoms followed on the part of the heart, muscles, and lungs, even going on to death from heart-failure. The author suggests the important and plausible explanation that in these cases partial immunity from previous infection exists in the body. This does not prevent the germs from developing, but does cause them to break down rapidly. Their toxic substances are thus set free, with the dangerous results now reported. Wasser-